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Связь между центральным аортальным давлением и диастолической функцией у пациентов, подвергающихся коронарной ангиографии

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Резюме

Актуальность. Колебания давления в аорте вносят существенный вклад в развитие атеросклероза, а ремоделирование сосудистой стенки, включая изменение артериальной жесткости, ассоциировано с сердечно-сосудистой заболеваемостью и смертностью. Колебания центрального артериального давления и его производных также влияют на ремоделирование миокарда и изменение его функции. **Цель исследования** — оценка взаимосвязей между уровнем центрального давления в аорте, измеренного инвазивным способом, и его производными, а также между состоянием диастолической функции левого желудочка (ЛЖ) и его скручиванием (торсией). **Материалы и методы.** В проспективное исследование было включено 90 пациентов с сохраненной функцией ЛЖ, направленных на выполнение коронарной ангиографии. **Результаты.** Выявлены значимые линейные взаимосвязи между эхокардиографическими показателями, отражающими диастолическую функцию ЛЖ, такими как индекс объема левого предсердия (ИОЛП), соотношение E/e’ ЛЖ (соотношение между скоростью раннего диастолического наполнения ЛЖ и ранней диастолической скоростью движения кольца митрального клапана), и центральным давлением в аорте. Поворот ЛЖ по оси (твист — от англ. twist) не коррелировал с показателями центрального давления в аорте. Только у 15 из 90 пациентов выявлена диастолическая дисфункция ЛЖ 2-й степени и выше со средним показателем поворота ЛЖ по оси 19,65 ± 9,4 против 16,70 ± 8,6 градуса при нормальном конечно-диастолическом давлении (p = 0,206). Это различие оказалось статистически значимым, отражая тот факт, что увеличение поворота ЛЖ по оси является доминирующим признаком у пациентов с высокой степенью диастолической дисфункции ЛЖ независимо от массы ЛЖ. **Заключение.** Мы установили взаимосвязь между центральным давлением в аорте и состоянием диастолической функции ЛЖ. Несмотря на нормальную фракцию выброса ЛЖ у всех обследованных, поворот ЛЖ по оси был увеличен у пациентов с диастолической дисфункцией ЛЖ 2-й степени и выше.

Ключевые слова: центральное давление в аорте, диастолическая дисфункция, скручивание левого желудочка, поворот по оси левого желудочка, торсия и твист левого желудочка, конечно-диастолическое давление левого желудочка

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Association between central aortic pressure and diastolic function in patients undergoing coronary angiography

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Abstract

Background. Aortic pressure dynamics significantly affect the development of arteriosclerosis, and chronic changes in the aortic wall, including surface stiffness, are strongly associated with cardiovascular mortality and morbidity. Variations in central aortic pressure and its derivatives have an impact on myocardial structural remodeling and function. This study **aimed** to determine the associations between invasively measured central aortic pressure and its derivatives and between left ventricular (LV) diastolic function status and torsion. **Design and methods.** This prospective study included 90 participants with normal LV function who were referred for elective coronary angiography. **Results.** There was a significant linear correlation between echocardiographic parameters reflecting LV diastolic function, such as the left atrial volume index (LAVI), the LV E/e' (ratio between mitral early diastolic velocity and early diastolic tissue annular velocity), and central aortic pressure. LV twist did not correlate with central aortic pressure parameters. Only 15 patients out of 90 had LV diastolic dysfunction of grade 2 and above, with a mean LV twist of 19,65 ± 9,4 vs 16,70 ± 8,6 degrees, as determined by normal LV end-diastolic pressure (p = 0,206). This difference appears clinically significant, illustrating that the increase in LV twist is predominant in patients with higher grades of LV diastolic dysfunction, irrespective of LV mass. **Conclusion.** There was a relationship and interaction between central aortic pressure and LV diastolic function status. Despite all participants had normal LV ejection fraction, LV twist appeared to be increased among the patients with LV diastolic dysfunction of grade 2 or above.

Key words: central aortic pressure, diastolic dysfunction, left ventricular torsion, left ventricular twist, left ventricular end diastolic pressure

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Introduction

Aortic stiffness is associated with increased cardiovascular morbidity and mortality, irrespective of traditional risk factors [1–3]. Noninvasive assessment of aortic stiffness is possible by tonometry, magnetic resonance imaging (MRI), and echocardiography. Increased aortic stiffness results in increased afterload, left ventricular (LV) hypertrophy, and altered coronary perfusion and, in extreme cases, may lead to diastolic heart failure [4, 5]. Echocardiographic demonstration of diastolic dysfunction and its association with invasive central aortic pressure will help identify the temporal effects of altered aortic stiffness on LV diastolic function. Central aortic blood pressure (CABP) is an important parameter that defines aortic flow dynamics and has a role in estimating stiffness. CABP has been proven to be a better predictor of adverse cardiovascular events than brachial artery pressure. CABP is also more strongly correlated with ventricular hypertrophy and carotid atherosclerosis than brachial pressure [4–7]. Increased central aortic pulse pressure (PP), fractional pulse pressure (FPP), and the pulsatility index (PI) independently correlate with reduced LV diastolic function status and elevated left ventricular end-diastolic pressure (LVEDP) [9]. Cardiac catheterization has proven to be the gold standard for central aortic pressure monitoring. Since it is an invasive technique, limited data have been obtained on the hemodynamic interactions of the LV and aorta. Hence, our study aimed to investigate whether there is any association between invasively measured CABP in patients who underwent invasive coronary angiography and LV diastolic function and LV twist by echocardiography.

Design and methods

This prospective observational study was carried out from January 2020 to May 2020. The institutional ethics committee approved the study protocol (IEC 794–2019 dated by 08/10/2019), and informed consent was obtained from all the participants. Consecutive patients who underwent an elective invasive coronary angiogram to evaluate coronary artery disease were included in the study after screening for exclusion criteria. Patients with unstable vital signs, ongoing chest pain, atrial fibrillation, significant valvular heart disease (greater than a mild degree of regurgitation or stenosis), past history of coronary artery disease, impaired LV systolic function (LV ejection fraction < 50 %), severe mitral annular calcification or left bundle branch block were excluded from the study. Detailed demographic parameters, laboratory findings, and a history of comorbidities were obtained from the recruited participants. Preprocedural electrocardiography (ECG) findings were recorded.

Echocardiographic assessment

Brief echocardiographic scanning was performed using a GE VIVID S60 echocardiographic machine (USA) with a 3–6 MHz transducer probe prior to the angiographic study. An experienced sonographer performed the test. Overall quantification of the left ventricle (LV) and left atrium (LA) was performed by acquiring apical 4-chamber and 2-chamber views. Conventional Doppler velocities across the atrioventricular and semilunar valves were attained. Early diastolic (E wave) and atrial contraction (A wave) velocities were obtained by applying pulsed-wave Doppler across the tip of the mitral valve (Fig. 1-A). The tissue annular velocity at the medial and lateral LV annulus was determined via tissue Doppler imaging (TDI) (Fig. 1-B). In addition, early diastolic (e’), late diastolic (a’), and systolic tissue annular velocities were measured across the medial and lateral LV annulus. Furthermore, LV twist and torsion, a measure of LV deformation, were assessed using speckle tracking echocardiography applied across the LV short-axis view at the basal and apical levels (Fig. 1-C). LV twist refers to the wringing motion of the left ventricle, defined as the net difference between basal and apical LV rotation. The torsion (in degrees/cm) is calculated as the twist divided by the longitudinal length of the LV. The aortic stiffness index was measured using the formula: [(SBP/DBP)] × [(AoS – AoD)/AoD], where SBP is the systolic blood pressure, DBP is the diastolic blood pressure, and AoS and AoD are the aortic dimensions in systole and diastole, respectively. In addition, the LV ejection fraction (EF), end-diastolic volume (EDV), and end-systolic volume (ESV) were measured.

Central aortic pressure monitoring during cardiac catheterization

Furthermore, during the elective coronary angiographic test performed by an interventional cardiologist, CABP was measured with the catheter placed in the aorta. The mean arterial pressure (MAP) was obtained by the formula: MAP = 1/3 systolic pressure + 2/3 diastolic pressure. Pulse pressure (PP = SBP – DBP), fractional pulse pressure (FPP = pulse pressure / aortic MAP), and the pulsatility index (PI = PP / aortic MAP) were obtained from the catheterization data.

Statistical analysis

The obtained data were analysed using SPSS version 16, where categorical variables are expressed as frequencies and percentages, whereas continuous variables are expressed as the mean ± standard deviation. Pearson’s correlation test was used to find a linear relationship between two continuous variables drawn from echocardiography and catheterization. An independent t-test was used to compare the continuous variables between two independent variables. A multivariable

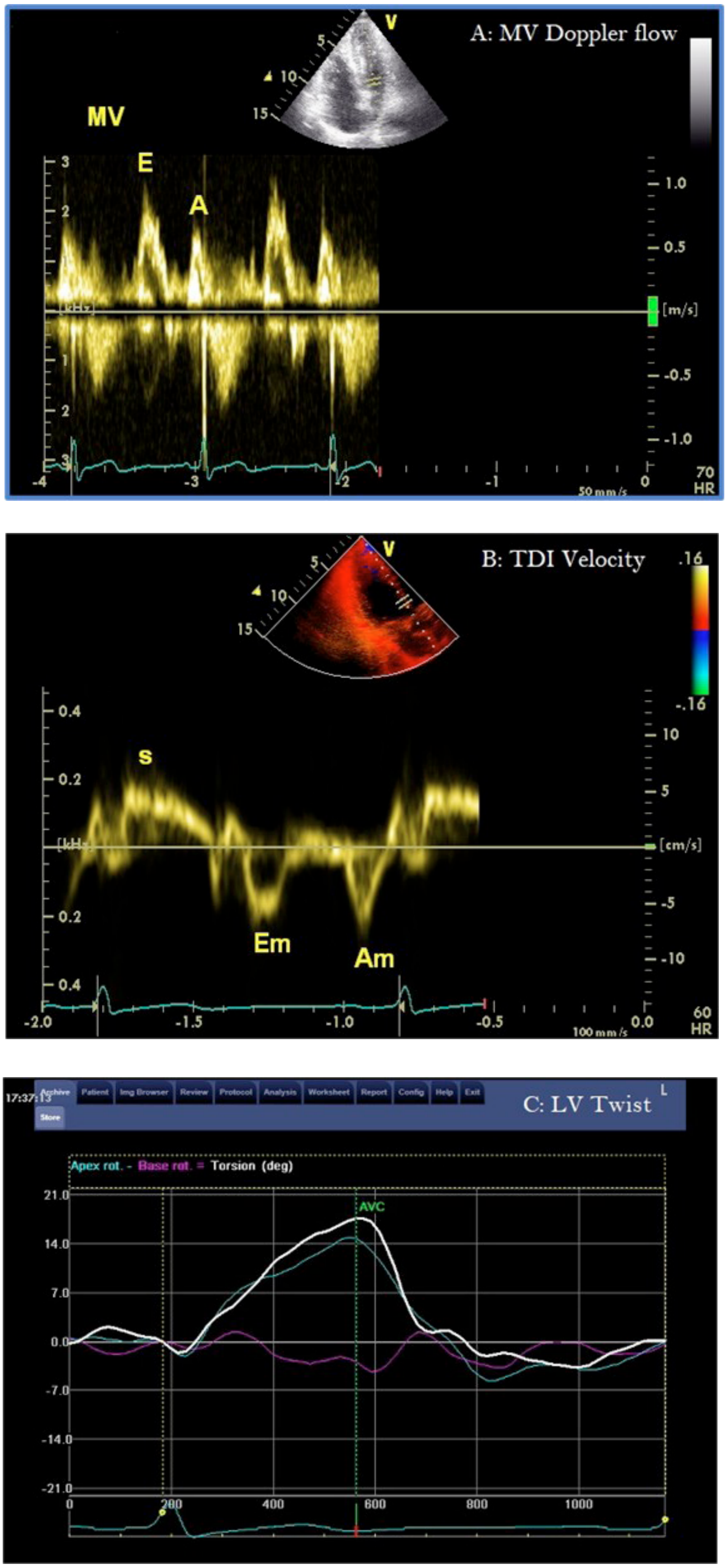

Figure 1. A: Mitral valve Doppler flow wave; B: Tissue Doppler imaging; C: Left ventricular twist assessment by tissue deformation imaging

Note: MV — mitral valve; E — early diastolic wave; A — atrial contraction wave; Em (e') — early diastolic tissue annular velocity; Am (a') — late diastolic tissue annular velocity during atrial contraction; S — systolic tissue annular velocity; LV — left ventricle.

regression analysis was performed in the prediction of LV E/e'. A p-value of < 0,05 was considered to indicate statistical significance.

Results

Baseline characteristics

The study included 90 subjects who underwent elective coronary artery angiography, among whom 60 (66,7 %) were males and 30 (33,3 %) were females; 40 (44,4 %) patients had a history of diabetes mellitus, 49 (54,4 %) patients were hypertensive, 9 (10,0 %) had a history of dyslipidemia, and 4 (4,4 %) were smokers. Tables 1 and 2 show the baseline demographic, clinical, laboratory, and echocardiographic data. Pearson's correlation test demonstrating the linear relationship between laboratory parameters and central aortic pressure derivatives showed that hemoglobin negatively correlated with the PI and FPP (r = −0,262, p = 0,014 and r = −0,275, p = 0,01, respectively). Among the lipid parameters, only low-density lipoprotein positively correlated with MAP (r = 0,292, p = 0,016), whereas total cholesterol, triglycerides, and high-density lipoprotein did not correlate with the CABP. Moreover, the glycated hemoglobin (HbA1c) and N-terminal pro brain natriuretic peptide (NT-pro-BNP) values did not correlate with the CABP derivatives.

Associations between central aortic pressure and diastolic function

Cardiac chamber quantification parameters such as LA and LV dimensions did not significantly correlate with CABP. However, the LV mass index (r = 0,297, p = 0,004) and relative wall thickness (RWT) (r = 0,225, p = 0,033) had good positive linear correlations with the

Table 1

PATIENT DEMOGRAPHIC, CLINICAL, AND LABORATORY DATA

Characteristics	Value (n = 90) [Mean ± SD or n (%)]
Age, years	58,7 ± 10,0
Male, n (%)	60 (66,7 %)
Body mass index, kg/m^2	24,93 ± 4,22
Brachial systolic blood pressure, mmHg	130 ± 19
Brachial diastolic blood pressure, mmHg	78,3 ± 9,5
Underlying medical conditions	
Hypertension, n (%)	49 (54,4 %)
Diabetes mellitus, n (%)	40 (44,4 %)
Dyslipidemia, n (%)	9 (10,0 %)
Current smoking, n (%)	4 (4,4 %)
Result of coronary angiography	
Insignificant stenosis, n (%)	18 (20 %)
1-vessel disease, n (%)	25 (27,8 %)
2-vessel disease, n (%)	17 (18,9 %)
3-vessel disease, n (%)	5 (5,6 %)
Laboratory findings	
Hemoglobin, g/dl	13,0 ± 1,4
HbA1c, %	7,5 ± 6,1
Total cholesterol, mg/dl	144 ± 44
Low density lipoprotein cholesterol, mg/dl	83 ± 36
High density lipoprotein cholesterol, mg/dl	43 ± 10
Triglyceride, mg/dl	146 ± 98

Table 2

ECHOCARDIOGRAPHIC ASSESSMENT OF LEFT VENTRICULAR SYSTOLIC AND DIASTOLIC FUNCTION AND CENTRAL AORTIC PRESSURE ASSESSMENT BY CARDIAC CATHETERIZATION

Parameter	Value (n = 90) (Mean ± SD)
Echocardiographic parameters	
Left ventricular ejection fraction, %	63,7 ± 9,4
Left ventricular end-diastolic dimension, mm	46,4 ± 6,8
Left ventricular mass index, g/m^2	119 ± 28
Relative wall thickness	0,46 ± 0,09
E wave velocity, cm/s	89,7 ± 10,2
A wave velocity, cm/s	91,2 ± 9,2
Deceleration time, ms	214 ± 59
E/A	1,01 ± 0,32
e’ velocity, cm/s	10,3 ± 1,0
a’ velocity, cm/s	9,2 ± 2,0
E/e’	11,0 ± 5,2
Left atrial volume index, ml/m^2	28,4 ± 9,5
Parameters of central aortic pressure monitoring by cardiac catheterization	
Systolic blood pressure, mmHg	130 ± 19
Diastolic blood pressure, mmHg	78 ± 9
Mean arterial blood pressure, mmHg	111,5 ± 15,6
Heart rate, bpm	75,8 ± 15,5
Pulse pressure, mmHg	73,0 ± 23,2
Fractional pulse pressure	0,65 ± 0,18
Pulsatility index	0,92 ± 0,36

MAP (Table 3). Diastolic function parameters, including LA volume index, mitral Doppler flow parameters, and tissue annular velocities, showed statistically significant linear correlations with the PI, FPP, and MAP. Among these variables, the LV E/e’ ratio significantly correlated with all the CABP derivatives, specifically the PI ($r = 0{,}242$, $p = 0{,}026$), FPP ($r = 0{,}251$, $p = 0{,}021$) and MAP ($r = 0{,}243$, $p = 0{,}025$) (Table 4).

Subgroup analysis between patients with LV E/e’ ≥ 15 and those with LV E/e’ < 15

Further subgroup analysis was carried out to compare CABP and LV diastolic function parameters between patients with high-grade (≥ grade II) LV diastolic dysfunction and patients with normal/grade I LV diastolic dysfunction. The study participants were categorized into two groups based on E/e’ values ≥ 15 and < 15. The E/e’ ratio is a load-independent parameter commonly used to estimate LV end-diastolic pressure (LVEDP). 15 patients had evidence of elevated LVEDP, denoted as an LV E/e’ ≥ 15, and 75 patients were included in the normal LVEDP group based on an LV E/e’ < 15. Analysis using an independent t-test showed that there was a significantly higher MAP among patients with elevated LVEDP, suggesting the effect of an increase in the MAP on the LVEDP and LV diastolic function (122,17 ± 15,85 vs 109,92 ± 15,40 mmHg, $p = 0{,}013$). Despite having only 15 patients with high grades of LV diastolic dysfunction, echocardiographic parameters of LV diastolic function correlated with CABP. This divulges the importance of echocardiography in the non-invasive assessment of LV remodelling and diastolic function status among patients with normal and increased blood pressure.

Table 3

CORRELATION COEFFICIENT IN THE ASSESSMENT OF LINEAR RELATIONSHIP BETWEEN DIASTOLIC FUNCTION PARAMETERS AND CENTRAL AORTIC PRESSURE DERIVATIVES

Parameters		PI	FPP	MAP
Body mass index, kg/m^2	r	–0,144	–0,121	–0,042
	p	0,177	0,256	0,694
Left atrium anteroposterior dimension, mm	R	–0,042	–0,045	–0,055
	p	0,691	0,673	0,610
Left atrium inferior-superior dimension, mm	r	–0,038	–0,037	–0,202
	p	0,719	0,727	0,056
Left ventricular end-diastolic dimension, mm	r	–0,075	–0,072	–0,172
	p	0,484	0,500	0,106
Left ventricular end-systolic dimension, mm	r	–0,097	–0,105	–0,088
	p	0,363	0,324	0,411
Interventricular septal thickness, mm	r	0,005	0,021	0,139
	p	0,960	0,842	0,191
Posterior wall thickness, mm	r	0,025	0,041	0,130
	p	0,812	0,703	0,222
Left ventricular mass, g	r	–0,049	–0,027	0,081
	p	0,648	0,802	0,448
Left ventricular mass index, g/m^2	r	0,058	0,086	0,168
	p	0,586	0,423	0,113
Ascending aorta in systole, mm	r	–0,078	–0,098	0,142
	p	0,505	0,403	0,223
Ascending aorta in diastole, mm	r	–0,101	–0,105	0,157
	p	0,389	0,368	0,179
Left ventricular end-diastolic volume, ml	r	–0,121	–0,106	0,207
	p	0,255	0,322	0,051
Left ventricular end-systolic volume, ml	r	–0,138	–0,121	0,099
	p	0,196	0,257	0,353
Left ventricular ejection fraction, %	r	0,112	0,098	0,115
	p	0,295	0,358	0,281
Left ventricular end-diastolic volume indexed, ml/m^2	r	–0,044	–0,026	0,297*
	p	0,678	0,806	0,004
Left ventricular end-systolic volume indexed, ml/m^2	r	–0,082	–0,063	0,164
	p	0,440	0,552	0,122
Relative wall thickness	r	–0,057	–0,059	0,225*
	p	0,596	0,581	0,033

Note: r — correlation coefficient, *: $p < 0,05$; PI — pulsatility index; FPP — fractional pulse pressure; MAP — mean arterial pressure.

Table 4

CORRELATION COEFFICIENT IN THE ASSESSMENT OF LINEAR RELATIONSHIP BETWEEN DIASTOLIC FUNCTION PARAMETERS AND CENTRAL AORTIC PRESSURE DERIVATIVES

Parameters		PI	FPP	MAP
Mitral E (early diastolic) wave, cm/s	r	0,020	0,028	–0,007
	p	0,852	0,796	0,952
Mitral A (late diastolic) wave, cm/s	r	0,038	0,045	0,011
	p	0,728	0,682	0,919
Deceleration time, ms	r	0,086	0,111	–0,302*
	p	0,434	0,313	0,005
Mitral E/A	r	–0,045	–0,042	–0,095
	p	0,680	0,700	0,390
e' (early diastolic tissue annular velocity) at IVS, cm/s	r	–0,109	–0,107	0,207*
	p	0,308	0,314	0,050
a' (late diastolic tissue annular velocity) at IVS, cm/s	r	–0,230*	–0,239*	–0,165
	p	0,029	0,023	0,120
e' (early diastolic tissue annular velocity) at LW, cm/s	r	–0,198	–0,188	–0,253*
	p	0,062	0,075	0,016
a' (late diastolic tissue annular velocity) at LW, cm/s	r	–0,237*	–0,243*	–0,018
	p	0,025	0,021	0,866
E/e'	r	0,242*	0,251*	0,243*
	p	0,026	0,021	0,025
Left atrial volume in 4ch view, ml	r	0,125	0,127	–0,016
	p	0,247	0,237	0,880
Left atrial volume in 2ch view, ml	r	0,162	0,173	–0,007
	p	0,129	0,105	0,948
Left atrial volume indexed, ml/m^2	r	0,228*	0,239*	0,026
	p	0,031	0,024	0,810
Left ventricular twist, ml/m^2	r	0,145	0,153	0,069
	p	0,190	0,168	0,535

Note: r — correlation coefficient, * — p < 0,05; IVS — interventricular septum; FPP — fractional pulse pressure; LW — lateral wall; MAP — mean arterial pressure; PI — pulsatility index; 2Ch — 2-chamber view; 4Ch — 4-chamber view.

The mean LV mass index was significantly greater among patients with elevated LVEDP (131,08 ± 33,20 vs 118,52 ± 27,93 g/m^2, p = 0,163) (Table 5). Since there were only 15 patients with elevated LVEDP, we could not achieve the required power, although Pearson's correlation test demonstrated linear association between an invasive measure of aortic pressure and non-invasive estimation of LV diastolic function. Furthermore, multivariate analysis showed that only MAP was independently associated with the LV E/e' ratio, whereas FPP and PI were not (Table 6).

Torsion and diastolic function association

LV twist, a measure of LV deformation, is an objective method of assessing ventricular function and showed increased values among patients with an LV E/e' ≥ 15. This finding depicts the compensatory mechanism in the form of increased myocardial stress in re-

Table 5

INDEPENDENT T TEST FOR COMPARISONS BETWEEN PATIENTS WITH NORMAL AND ELEVATED LV END-DIASTOLIC PRESSURE

Parameters	**E/e' < 15 Mean ± SD N = 75**	**E/e' ≥ 15 Mean ± SD N = 15**	**p-value**
MAP, mmHg	109,92 ± 15,40	122,17 ± 15,85	0,013
FPP, mmHg	0,65 ± 0,18	0,68 ± 0,23	0,718
PI	0,93 ± 0,36	0,98 ± 0,40	0,693
LV twist, degrees	16,70 ± 8,6	19,65 ± 9,40	0,206
LV mass index, g/m^2	118,52 ± 27,93	131,08 ± 33,20	0,163
LV mass, g	199,48 ± 51,08	208,50 ± 57,59	0,582
EF, %	64,05 ± 9,92	61,67 ± 7,85	0,430

Note: E — mitral early diastolic velocity; e' — early diastolic tissue annular velocity; EF — ejection fraction; FPP — fractional pulse pressure; LV — left ventricle; MAP — mean arterial pressure; PI — pulsatility index.

Table 6

MULTIVARIABLE ANALYSIS FOR THE PREDICTION OF LEFT VENTRICULAR E/e'

Model	**Unstandardized coefficients**		**Standardized coefficients**	**t**	**p-value**	**95 % confidence interval for B**	
	B	**Std. error**	**Beta**			**Lower bound**	**Upper bound**
MAP	0,079	0,034	0,241	2,316	0,023	0,011	0,146
FPP	9,707	14,080	0,353	0,689	0,493	–18,307	37,722
PI	–1,522	7,282	–0,107	–0,209	0,835	–16,012	12,967

Note: FPP — fractional pulse pressure; MAP — mean arterial pressure; PI — pulsatility index.

sponse to elevated LVEDP. Although the result was not statistically significant, this alteration appears to have clinical significance (19,65 ± 9,40 vs 16,70 ± 8,6 degrees, p = 0,206) (Table 5).

Discussion

The present study illustrates that there is a strong correlation between diastolic function parameters, as assessed by echocardiography, and invasively measured CABP. Specifically, diastolic function parameters obtained by Doppler echocardiography, tissue Doppler imaging (including LV E/e'), and the LA volume index showed good linear correlations with CABP derivatives such as the PI, FPP, and MAP. Although approximately 49 (54,4 %) patients were hypertensive, only 15 (16,66 %) had elevated LVEDP (LV E/e' value ≥ 15). Sub-analysis showed significant elevation in the mean arterial pressure, LV mass, and LV twist among patients with elevated LVEDP; however, these differences were not statistically significant. We report a clinically significant difference in LV twist and LV mass between patients with normal and elevated LVEDP. Our study demonstrated that LV E/e' was independently associated with the MAP only among all CABP derivatives. In a similar study, CABP parameters were independently associated with the LV e' and E/e' values [8]. A comparable study with a sample size of 104 patients showed correlation between invasively measured LVEDP and CABP [9]. However, in our study, we demonstrated that noninvasively examined echocardiographic LVEDP parameters correlate well with central aortic pressure among patients enrolled for invasive coronary angiography. In another study focusing on gender predilection in the association between LV diastolic function parameters and invasively measured aortic pulse pressure, a significant correlation between E/e' and pulse pressure was found only among elderly women but not in men [10]. A study specifically investigating heart failure with a preserved ejection fraction showed a significant association between the augmentation index and LV diastolic dysfunction status and between the augmentation index and NT-pro-

BNP levels. Thus, the study postulated the importance of an altered augmentation index in the hemodynamic changes in LVEDP and heart failure development [11]. Moreover, a study conducted by S. Romagnoli et al. (2014) demonstrated that invasive aortic pressure measures could be inaccurate due to artefacts created by damping or resonance [12].

Our study demonstrated a strong association of central PP, FPP, MAP, and PI with elevated LV filling pressure and LA volume index. Increased MAP and reduced LV relaxation status also significantly correlated. No mechanism in the pathways associated with altered aortic stiffness resulting in impaired LV relaxation has been defined yet. In the proposed mechanism, a stiffened aorta augments the systolic BP and reduces diastolic BP, consequently increasing PP [13, 14]. In hypertension, elevation in afterload, which affects normal contraction, and a reduction in coronary reperfusion, which disturbs LV relaxation, act as the basic element for LV remodeling, resulting in hypertrophy. In addition, increased aortic stiffness is also a contributing factor to the development of subendocardial ischemia, which adversely precipitates LV diastolic dysfunction [9, 15, 16]. Furthermore, CABP and its derivatives have proven to be the best predictors of cardiovascular episodes and even deaths compared to peripheral aortic pressure variables [17–19]. Our study attempted to evaluate and verify the relationships between CABP and LV diastolic function. Tissue Doppler imaging parameters such as e' and E/e' are vital for assessing LV diastolic dysfunction and strongly correlate with CABP. This indicates that using tissue Doppler imaging in routine practice supports the decisive diagnosis of LV diastolic dysfunction and has been proven to be the best marker for assessing LV diastolic dysfunction [20–23]. Nevertheless, TDI-derived parameters also showed prognostic implications for cardiac patients [24–26]. In addition, the present study investigated the use of LV twist in LV diastolic function assessment. The results showed a significant increase in twist among patients with elevated LV EDP. However, the sample size was too small to generate the required power.

LV twist has been extensively studied, focusing on assessing subtle LV diastolic and systolic dysfunction. Compared with healthy controls, patients with hypertrophic cardiomyopathy exhibit an increase in twist and torsion [27]. Additionally, studies demonstrated that the severity of LV diastolic dysfunction is directly proportional to LV twist. However, the change is more obvious among patients with a higher grade of LV diastolic dysfunction [28, 29]. This demonstrates cardiac deformational changes in response to reduced compliance. The present study consisted of only 15 patients with higher grades of LV diastolic dysfunction, and it did not correlate with CABP, but it had a higher mean value among patients with higher grades of LV diastolic dysfunction.

A few limitations of the current study warrant discussion. The possibility of a temporal relationship between LV diastolic dysfunction and CABP cannot be excluded because of the use of cross-sectional data. Second, this was a single-center Indian population study, so the results may not be applicable to another population. There were fewer participants with diastolic dysfunction of grade II or greater (15 out of 90). Finally, half of the patients were hypertensive and received vasoactive drugs that alter hemodynamics, which might have caused selection bias and could limit the generalizability of our findings. In the future, assessing all grades of LV diastolic dysfunction and their relationship with CABP and subclinical LV remodeling changes, as proven by deformation imaging (using LV twist), will increase the scope of new budding research.

Conclusion

The study demonstrated a significant correlation between LV diastolic function parameters and CABP. This elucidates a relation and interaction between CABP and the stiffness index with LV diastolic function status. LV E/e' and LAVI were the best echocardiographic measures that correlated with the PI, FPP, and MAP. Despite having a normal LV ejection fraction among all the participants, LV twist appeared to be increased among the patients with LV diastolic dysfunction of grade 2 or above. Forthcoming work on assessing subclinical LV remodeling among patients with a lower grade of LV diastolic dysfunction along with CABP derivatives may enhance the scope of future research in this area.

Ethics approval statement & clinical trial registration

The study was approved by the institutional ethics committee and registered in the Clinical trial registry of India (CTRI/2020/01/022756 dated 14/01/2020).

Consent form

Consent was taken from all patients before collecting the information and manuscript preparation.

Conflict of interest / Конфликт интересов

Authors declare no conflict of interest. / Авторы заявили об отсутствии конфликта интересов.

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